HELPFUL INFORMATION:

SMOKING & HEALTH

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Kensler, C. J., "Components of Pharmacologic Interest in Tobacco Smoke," Ann. N. Y. Acad. Sci. 90(1): 43-47, 1960.

> "In conclusion, it should be pointed out that estimation of pharmacological importance of the amounts of the compounds present in tobacco smoke is best related to the response of the average individual, and should not be generalized to include individuals who are extremely senstitive because of genetic or other reasons, or to those individuals who, as a result of differential aging or the presence or history of disease, may differ markedly from the average or normal in their response."
>
> p. 46-47

Bentley, H. R. & J. G. Burgan, "Cigarette Smoke Condensate: Preparation and Routine Laboratory Estimation," Tob. Man. Stand. Comm. (4): 2-9, 1961.

> "The main conclusions to be drawn from the above experimental results are as follows. . .The same brand of cigarettes made at different times can have different yields of smoke condensate. The yield of smoke condensate from a cigarette varies with the conditions under which the cigarette is smoked."
>
> p. 8

Reddy, D. G., et al., "Experimental Production of Cancer with Cigarette Tar," Indian Journal Medical Research 57(1): 125-127, 1969.

> "Our findings in these two experiments show that tobacco and cigarette smoke condensate are not by themselves complete carcinogens."
>
> p. 126

Davies, R. F. & J. K. Whitehead, "A Study of the Effects of Altering the Tar/Nicotine Ratio in Experimental Tobacco Carcinogenesis," British Journal Cancer 24(1): 191-194, March, 1970.

> "There was no statistically significnat difference in specific mouse skin carcinogenicity between smoke condensate from plain, flue-cured tobacco cigarettes with a normal tar to nicotine ratio and condensate from filter-tip cigarettes made from selected flue-cured tobaccos with a reduced tar to nicotine ratio."
>
> p. 191

Okun, R., Testimony, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U. S. Senate, February 1, 3 & 10, 1972, pp. 230-234. (SH)

> " . . . a recent study has failed to show any statistically significant differences in animal effects between tar from one type of cigarette with a normal tar-nicotine content and the tar from filter-tipped cigarettes made from other types of tobacco with reduced tar-nicotine."
> p. 230

> "From my experience and review of the scientific literature and the recent Surgeon General's report, I cannot tell the significance of nicotine content, tar content, and potential carbon monoxide levels derived from cigarette smoke on health.
>
> "However, a false sense of significance, if not security, can be derived by the nonscientific public from government establishment or approval of maximum levels of these constituents."
> p. 232
>
> "There are many things that bear on tar content of a cigarette that have to do with more than just the grams of tobacco that exist in a cigarette:
>
> > "The type of tobacco, where the tobacco is grown on the plant and whether sheets are used, stems are used, et cetera, or whether it is chemically treated.
>
> "All these things bear on the quality of tar. You may find that the weight of tar between a cigarette is the same, but qualitatively quite different."
> pp. 233-234

Soloff, L. A., Statement, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U.S. Senate, February 1, 3, & 10, 1972 (as quoted by Senator Marlow Cook, Closing Statement, p. 286).

> "In conclusion, the data linking heart disease and smoking are still questionable. Studies with particular reference to twins in the United States and Sweden and epidemiological studies in many countries fail to show a correlation between smoking and coronary heart disease. Human studies on smoking that utilize highly artificial circumstances are of questionable validity. Such data do not provide support for the proposed arbitrary limitation on so-called 'incriminated agents' in cigarette smoke."

Rigdon, R. H., Statement, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U.S. Senate, February 1, 3, & 10, 1972 (as quoted by Senator Marlow Cook, Closing Statement, p. 286).

> ". . .to my knowledge, it has never been scientifically established that tobacco smoke condensate is harmful or hazardous to smokers.
>
> ". . . my experiments emphasize the fact that much additional information is needed before we draw any conclusions about the effects of tobacco smoke condensate or any of its constituents on smokers."

Furst, A., Testimony, Hearings before the Consumer Subcommittee of the Committee of Commerce, U.S. Senate, February 1, 3, & 10, 1972, pp. 203-217. (SH)

> ". . .setting limits on compounds formed in tobacco smoke or condensate would be to act in what is nearly a scientific vacuum as far as good experimental data is concerned." p. 207
>
> "Condensates will vary depending on the conditions of burning and on the composition of cigarettes; unless you absolutely specify every single factor, moisture content, tobacco content, burning zone, rate of burning, you will get different condensates." p. 215

Ashton, H. & R. Telford, "Smoking and Carboxyhaemoglobin," The Lancet pp. 857-858, October 13, 1973.

> "To publish the CO yield as well as the tar and nicotine content of different brands of cigarettes without qualification. . .could be misleading. Our results indicate that smokers smoking without constraint actually obtain more CO (and much the same amount of nicotine) from low-nicotine (mild) cigarettes than from high-nicotine (non-mild) cigarettes."
>
> p. 857

Ashton, H. & R. Telford, "Blood Carboxyhaemoglobin Levels in Smokers," British Medical Journal 4: 740, December 22, 1973.

> "The rise in COHb during smoking was greatest for the low- and least for the high-nicotine cigarettes. . . . This difference was accompanied by a tendency towards a lower puffing rate for the high-nicotine cigarettes, though more nicotine was in fact delivered. Thus the subjects were able to obtain a relatively high dose of nicotine with a smaller increase in COHb from the high- as compared with the low-nicotine cigarettes."

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag (Stockholm 1973) pp. 341-362.

> "The animal models used to study the development of arteriosclerosis, and in particular coronary arteriosclerosis, ischaemic heart disease and myocardial infarction are not representative of the human disease. Consequently, though much work has been invested in these animal experiments few conclusions can be drawn from the results of these to the clinical or human situation. This is especially true for the huge literature on dietary studies using maximally distorted diets with fats, carbohydrates, proteins and vitamins in nonrealistic relations when compared with ordinary clinical situations. This is also true for animal studies on behaviour, cigarette smoking (or influence of nicotine or of carbon monoxide)."
>
> p. 350

Russell, M. A. H., et al., "Plasma Nicotine Levels After Smoking Cigarettes With High, Medium, and Low Nicotine Yields," British Medical Journal, pp. 414-416, May 24, 1975.

> "The findings suggest that the plasma nicotine level just after a cigarette depends more on the way the cigarette is smoked than on its nicotine yield or the number which have been smoked over the preceding few hours."
>
> p. 414

Eskwith, I.S., "Etiology of Atherosclerotic Heart Disease," American Heart Journal 90(6): 809-810, December, 1975.

> "So far no one has found any direct effect of nicotine on the heart other than it somewhat increases cardiac output. Cardiologists today are sending poor middle-aged men out on the road, exercising, to achieve therefore, the same results that could be obtained by a half a package of cigarettes."
>
> p. 809

Ito, H. & D.M. Aviado, "Pulmonary Emphysema and Cigarette Smoke - Experimental Induction and Use of Bronchodilators in Rats," Archives of Environmental Health 16(6): 867-870, June, 1968.

> "The results are of particular interest because they question the validity of the widely accepted belief that cigarette smoke can promote the development of pulmonary emphysema."
>
> p. 865

Bauer, D.R., Medical World News 10: 34, August 22, 1969.

> "When Medical World News embarked on the current environmental health series. . .we wanted to photograph two lungs: one taken from a mature adult who had lived his life in the country and the other from an urban dweller. There was no difficulty in locating a predictable city lung blackened by years of inhaling polluted air. . . .
>
> "The search for a clean lung led south to Virginia and, finally, to Vermont. Both the lungs there were all discolored. Even amid the bucolic splendors of Vermont, reports Dr. Lawrence S. Harris, that state's chief medical examiner, it is increasingly difficult to find a clean lung in a mature adult. Only by photographing the lung of a four-month-old infant who died of epilepsy did we find the expected contrast."

Levine, E. R., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 1334-1339.

> "I believe that emphysema must be listed among the vascular diseases. As such, I cannot find any actual evidence that anything inhaled into the bronchial tree, cigarette smoke or anything else, has a causal relationship to the development of this disease."
>
> p. 1336

Roberts, K. E., "Emphysema and Enterobacterial Infection," Medical Counterpoint 2(3): 47, March 1970.

> "In humans, only two lines of evidence have linked cigarette smoking and emphysema; one is statistical and the other is political. . . .The reasonable doubts of the minority are cultured by this study and require another trial in the court of facts before assigning emphysema to man's external rather than internal environment."

Rosenblatt, M. B., "Validity of Emphysema Mortality Data," Review of Allergy 24: 1019-1022, November 1970.

> "Quantophrenia, the state of fanatic obsession with numbers, is a common affliction among epidemiologists often leading to unwarranted conclusions. . . . If our current breed of statisticians would take the time to study the history of emphysema during the past 300 years they would not be so zealous in implicating cigarettes as an etiologic agent."
>
> p. 1020

Aviado, D. M., et al., "Cigarette Smoke and Pulmonary Emphysema," Archives of Environmental Health 20: 483-487, April 1970.

> "The experiments in rats have failed to support the widespread belief that cigarette smoke can induce experimental pulmonary emphysema."
>
> p. 487

Rosenblatt, M. B., "Emphysema: Historial Persective," Bulletin of N.Y. Academy of Medicine 48(6): 823-841, July 1972. (SH)

> "It is difficult to reconcile the prevalence of emphysema in the 19th century with official statistics showing virtual nonexistence of the disease in the early decades of the 20th century. The present review is not concerned with the reasons for this disparity except to point out that the sudden apparent increase of emphysema in the United States may well be an artifact produced by revision of the International List of the Causes of Death in 1949 permitting emphysema, for the first time, to be accepted as a primary cause of death."
>
> p. 838

Dimond, J. L., "Chronic Bronchitis," New Zealand Medical Journal 75: 38, January, 1972. (TI) (SH)

> "Within the next decade English physicians will probably be agreeing with their Continental colleagues that chronic bronchitis is in actual fact a psycho-physiological disorder.
>
> ". . . although the rate of smoking per adult has increased threefold in the last 50 years there has been no increased incidence in standard mortality rates from bronchitis in males. . . ."

Sherman, J., et al., "A Health Research Group Study on Disease Among Workers in the Auto Industry," Washington, D.C.: Unpublished Paper Released September 7, 1973, p. 11. (SH)

> "These data . . . seriously challenge the traditional view by management and much of the medical profession that workers' lung and heart diseases are largely caused by cigarettes rather than by workplace poisons."
>
> p. 11

National Heart & Lung Institute, Submission, Hearings Before a Subcommittee of the Committee on Appropriations, House of Representatives, March 26, 1974, p. 281.

> "But we do not know the cause of pulmonary emphysema, how to stop its progress even if detected early, or how to prevent heart disease caused by emphysema."

Walker, W. J., "Headline Correction," Medical Tribune, March 26, 1975, p. 11.

> "The age-adjusted death rate from emphysema peaked in the United States in 1968 and has declined each year since then. . . .Instead of the fastest growing cause of death it turns out to be a 17% decline during a five year period!"

Blot, W.J. & J.F. Fraumeni, "Arsenical Air Pollution and Lung Cancer," The Lancet, pp. 142-144, July 26, 1975.

> "The most likely explanation for the increased lung-cancer mortality in this study is neighbourhood air pollution from industrial sources of inorganic arsenic.
>
> "The carcinogenic effect of asbestos is not confined to workers, but may extend to surrounding neighbourhoods and families: our findings, although preliminary, suggest that the carcinogenic hazard of arsenic may cross plant boundaries into the general community.
>
> "It is possible that tobacco consumption is greater in counties with copper, lead, or zinc industries, but there is no reason to suspect this. The smoking habits of workers in copper smelters were not unusual."
> p. 144

Brown, S. M., et al., "Effect on Mortality of the 1974 Fuel Crisis," Nature 257: 306-307, September 25, 1975.

> "The 1974 fuel crisis was a natural experiment. It presented the opportunity to test the hypothesis that a decrease in vehicular exhaust fumes would have a beneficial effect on health. . . . Dramatic decreases were noted in death rates for several major categories of disease. . . .The disease showing the greatest relative change was chronic lung disease."
>
> p. 306

Fifer, W. R., "Air Pollution - A Primer For Practicing Physicians," Bulletin of the St. Louis Park Medical Center 13(3): 1-7, 1969.

> "There is mounting evidence that the serious chronic lung diseases such as lung cancer, chronic bronchitis and emphysema may result, at least in part, from air pollution."
> p. 6

McCall, M. H. & N. S. Stenhouse, "Deaths from Lung Cancer in Australia," The Medical Journal of Australia, pp. 524-525, March 6, 1971.

> "The effect of an environmental agent such as air pollution would be strongly supported by evidence that the death rate from lung cancer in British immigrants increases with increasing periods of residence in England before immigration to Australia. . . .Since smoking habits do not vary greatly between the countries studied, the findings reported here strongly support Dean's conclusion that the role of air pollution in the genesis of lung cancer has been seriously underestimated."
> p. 525

Eisenbud, M., & L. R. Ehrlich, "Carbon Monoxide Concentration Trends in Urban Atmospheres," Science, pp. 193-194, April 14, 1972. (SH)

> "The predominant modern source of CO is the exhaust gases from the internal-combustion engine. Approximately 97 percent of the CO in the air of New York City is currently attributable to this source."
>
> p. 193

"Work in America", Report of a Special Task Force to the Secretary of Health, Education and Welfare, December, 1972. (SH)

> "In addition to heart disease, several other illnesses have been found to be highly associated with occupational stress. The most convincing evidence pertains to peptic ulcers, and both arthritis and rheumatoid arthritis. Links also have been suggested for stroke and gout (a form of arthritis)."
>
> p. 66

Lave, B. & E.P. Seskin, P., "Air Pollution, Climate, and Home Heating: Their Effects on U. S. Mortality Rates," American Journal of Public Health 62: 909-916, July, 1972. (SH)

> "These studies make it apparent that there is a close association between mortality rates and air pollution. This investigation strengthens the conclusions cited in a previous work that mortality rates could be lowered substantially by abating air pollution."
>
> p. 915

Sterling, T. D., "Difficulties of Measuring the Effects of Air Pollution vs. Effects of Smoking," Paper Presented Before the American Association for the Advancement of Science, December 27, 1972, Washington, D. C. (SH)

> "A study of the air pollution and smoking effects inevitably then reaches two conclusions.
> 1. At a technical level, existing evidence is derived from inadequate studies. . . .
> 2. A pall of secrecy hangs over much of the crucial information that is needed to form valid conclusions about health effects either of smoking or of occupational and community pollution."
>
> pp. 18-19

Hueper, W.C., "Medicolegal Considerations of Occupational and Nonoccupational Environmental Cancer," Chapter 38 (VII) in: Lawyers Medical Cyclopedia of Personal Injuries and Allied Specialties (Revised Vol. 5, Part B), Frankel, C.J. (Editor), The Allen Smith Co., (Indianapolis, 1972), pp. 301-302. (SH)

> "Occupational carcinogenic agents encountered during regular occupational activities furnish the majority of known environmental carcinogens. . . .While many millions of workers, probably even the majority, have some contact with one or several of such agents, carcinogenic occupational hazards and the occurrence of occupational cancers have been established by epidemiologic studies for only a comparatively small fraction of the exposed working population, because neither industry nor governmental public health agencies have been interested sufficiently to determine the presence and scope of cancer hazards among the bulk of workers exposed."

Sterling, T.D., "Air Pollution and Smoking," Environment 15(6): 3-5, 25-26, July/August, 1973. (TI)(SH)

"However, in the enthusiasm of many of our colleagues in attacking cigarette smoking as the major public health problem, the effect of cigarette smoking has been increasingly contrasted with that of pollutants and industrial exposure until today a real question exists if cigarette smoking is not diverting attention from the effects of occupational exposure on industrial workers and of the increasing air pollution burden on the population of our communities."

p. 3

"The juxtaposition between smoking and the effects of industrial and community pollution are of considerable importance and need to be sanely reassessed. There is also the possibility that the fervor with which many environmentalists attack smoking will be used to protect present uses of organic fuels from pressures exerted on them by air pollution research findings."

p. 4

"To begin with, many specific lung diseases are known to be caused by particles in the industrial environment. . . .While the effects of these chemicals, working by themselves or together, are largely unexplored, it has been shown that many substances in the industrial and community environment that exist in significant quantities possess considerable carcinogenic potential."

p. 5

"Many studies have concluded outright that the major effect on pulmonary disease and lung cancer appears to be carried by environmental pollution and not by smoking."

p. 25

Stewart, R.D., et al., "Carboxyhemoglobin Concentrations in Blood From Donors in Chicago, Milwaukee, New York, and Los Angeles," Science 182: 1362-1364, December 28, 1973. (SH)

> "Among the nonsmokers, 76 percent in Los Angeles, 74 percent in Chicago, 35 percent in New York, and 26 percent in Milwaukee had COHb saturations in excess of 1.5 percent, indicating CO exposure in excess of that permitted by air quality standards."
>
> p. 1363

Sherman, J., et al., "A Health Research Group Study on Disease Among Workers in Auto Industry," Washington, D.C.: Unpublished Paper Released September 7, 1973, p. 11. (SH)

> "These data. . .seriously challenge the traditional view by management and much of the medical profession that workers' lung and heart diseases are largely caused by cigarettes rather than by workplace poisons."

Mancuso, T.F. & T.D. Sterling, "Lung Cancer Among Black and White Migrants in the U.S.," Journal of the National Medical Association 67(2): 106-112, March 1975. (SH)

> "Existing evidence does not support the smoking hypothesis. . . .In fact, only about half as many blacks smoked more than one pack a day as did whites (both in the North and South). . . .We think it reasonable to assume that smoking is completely unrelated to the difference in lung cancer between blacks and whites and migrants and non-migrants."
>
> p. 108

> "Implicated are some factors associated with place of birth in the U.S. and in particular in the South and the influences of early years of life and exposure to adverse industrial environments subsequent to migration. . . .Our findings support the hypothesis that lung cancer, at least among the blacks, may be largely a result of migration and occupational exposure to chemical dusts and fumes in the industrial employment."
>
> p. 111

Blot, W.J. & J.F. Fraumeni, "Arsenical Air Pollution and Lung Cancer," The Lancet, pp. 142-144, July 26, 1975.

> "The most likely explanation for the increased lung-cancer mortality in this study is neighbourhood air pollution from industrial sources of inorganic arsenic. . . .
>
> "The carcinogenic effect of asbestos is not confined to workers, but may extend to surrounding neighbourhoods and families: our findings, although preliminary, suggest that the carcinogenic hazard of arsenic may cross plant boundaries into the general community.
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> "The 1974 fuel crisis was a natural experiment. It presented the opportunity to test the hypothesis that a decrease in vehicular exhaust fumes would have a beneficial effect on health. . . . Dramatic decreases were noted in death rates for several major categories of disease. . . .The disease showing the greatest relative change was chronic lung disease."
>
> p. 306

Leshan, L.L. & R.E. Worthington, "Personality as a Factor in Pathogenesis of Cancer: A Review of the Literature," British Journal Medical Psychology XXIX(1): 49-56, 1956. (SH)

> "As one examines these papers, one is struck by the fact that there are consistent factors reported in studies which gathered their material in different ways. There appear to be four separate threads which run through the entire literature. These are: (1) the patients loss of an important relationship prior to the development of the tumour; (2) the cancer patient's inability successfully to express hostile feelings and emotions; (3) the cancer patient's unresolved tension concerning a parental figure; and (4) sexual disturbance."
>
> p. 54

Eysenck, H.J., Smoking, Health and Personality, Basic Books, (New York, 1965) 166 pp. (CTR) (SH)

> ". . . the evidence on the whole tends to support the view that constitutional factors in general and personality factors in particular are correlated with proneness to cancer."
>
> pp. 116-117

Kissen, D.M., "Possible Contribution of the Psychosomatic Approach to Prevention of Lung Cancer," Medical Officer, pp. 343-345, December 24, 1965. (SH)

> ". . . those with a poor outlet for emotional discharge appear to have more than four and a half times the mortality rate for lung cancer compared with those with a good outlet, and more than two and a half times the rate of those with a moderate outlet."
>
> pp. 343-344

Syme, S.L., "Is There a Future for the Epidemiologic Study of Coronary Heart Disease?" Paper Presented at the Meeting of the American Public Health Association, Detroit, November 11, 1968. (SH)

> "Seventh Day Adventists may have lower death rates from all causes for a number of reasons. It may be, for example, that SDA's constitute a highly selected group of people either in terms of genetics or of personality or of both."
> p. 4

Walter, E. & J.C. Walters, "Anxiety of Smoking and Nonsmoking Pregnant Women," Paper Presented at the Ninth Annual Meeting of the Society for Psychophysiological Research, October 16-19, 1969. (Abstract Only) (CTR)

> "Results of recent investigations into the psychological characteristics of smokers and nonsmokers suggests that the two differ in certain psychological traits. Eysenck (1960) had theorized that extraverts could be expected to smoke more cigarettes than introverts. . . . Eysenck (1965) postulated that extraverts suffer from a 'kind of stimulus hunger,' and thus have a need to partake of stimulating foods and drugs. His studies on male extraverts and introverts confirmed his hypothesis; Smith (1967) confirmed these results in females.
>
> "Comparison of the two groups [smokers & nonsmokers] on the Mann-Whitney U showed the smokers to have significantly higher anxiety scores than the nonsmokers."

Seltzer, C.C., Testimony, Hearings Before the Committee on Interstate & Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 531-544.

> "It has been suggested by many scientists that such an explanation may lie in the constitutional and genetic factors involved in coronary heart disease and in smoking behavior. That there is a strong genetic factor in the etiology of coronary heart disease is well accepted, and there is a growing body of evidence that smokers as a group differentiate themselves from nonsmokers in a large variety of biological ways including 'style of life.' If smokers develop coronary heart disease because they are different kinds of people than nonsmokers, more vulnerable constitutional types, this could well explain the statistical association of excess heart disease among cigarette smokers."
>
> p. 533

Jenkins, C.D., et al., "Association of Coronary-Prone Behavior Scores with Recurrence of Coronary Heart Disease," Journal of Chronic Disease 24(10: 601-610, 1971. (SH)

> "Evidence has been accumulating in recent years that social and psychological factors are involved in an important way with the etiology of coronary heart disease. . . .
>
> "This overt behavior patter (Type A) has been shown to be associated with increased prevalence of coronary heart disease (CHD) by three different research groups. . . ."
>
> p. 601

Burch, P.R.J., "Smoking and Cancer," Lancet 1: 939-940, April 28, 1973. (SH)

> "So far, I have been unable to devise any causal hypothesis of the effect of cigarette smoking that is clearly consistent with the epidemiological evidence for lung cancer: neither have I been able to falsify the constitutional hypothesis."
>
> p. 940

Rae, G. & J. McCall, "Some International Comparisons of Cancer Mortality Rates and Personality: A Brief Note," The Journal of Psychology 85: 87-88, 1973. (SH)

> "The positive correlations obtained between extraversion and lung cancer rates in both males and females and the negative correlations between these rates and anxiety lend tentative support, on a cross-national basis, to Eysenck's postulate that 'persons constitutionally predisposed to take up smoking are also constitutionally predisposed to develop cancer.'"
>
> p. 88

Coan, R.W., "Personality Varibles Associated with Cigarette Smoking," Journal of Personality and Social Psychology 26(1): 86-104, May 1973. (TI) (SH)

> "Obviously it behooves us as scientists to recognize that the well-substantiated correlation between smoking and lung cancer is not definitive evidence that the former causes the latter. . . . smoking is but one symptom of a pattern of living that is generally hazardous. Thus, the cancer could be a consequence of varied stresses associated with smoking, not a consequence of smoking itself."
>
> p. 103

Hickey, R.J., et al., "Aryl Hydrocarbons, Smoking and Lung Cancer," New England Journal of Medicine 390(10): 576-577, March 7, 1974. (SH)

> "The association of cigarette smoking behavior with lung cancer risk is well known, but inference of causality from correlation is invalid. Moreover, ecologically acceptable animal studies have generally failed to support the hypothesis that cigarette smoking causes lung cancer. An alternative hypothesis, that cigarette smoking and lung cancer risk are influenced by a common cause - the individual constitution or genotype - appears compatible with observed data. Smoking may be symptomatic of constitutional deficiencies that render smokers, on the average, more vulnerable than nonsmokers to damaging effects of air pollutants."

Burch, P.R.J., "Problems in the Interpretation of Cancer Statistics With Special Reference to Lung Cancer," Journal Society of Occupational Medicine 25: 2-10, 1975.

> "My analysis. . .indicates that most human cancers, as recorded in national mortality statistics, are spontaneous in origin. I infer that they arise, in genetically predisposed persons, as the result of the intrinsic instability of genes in stem cells of the central system of growth-control."
>
> p. 9

> "The largest study available so far of mortality in twins appreciably discordant for smoking habits is that of Friberg et al. (1973), carried out in Sweden. Among 572 discordant pairs of MZ [monozygotic] twins, 31 'first' deaths had been recorded among non- and 'low'- smokers and 32 among the 'high'-smoking group. The equality of death-rates among the 'low' and 'high' smoking members of discordant MZ pairs agrees with the expectations of the constitutional hypothesis and conflicts with the causal hypothesis."
>
> p. 9

Selye, H., "Smoking Introductory Remarks," Chapter 1 in: Smoking Behavior: Motives and Incentives, W. L. Dunn Ed., V. H. Winston & Sons (Washington, D. C., 1973), 309 pp.

> "The choice is not 'to smoke or not to smoke,' but whether to smoke, or to overeat, to drink, to drive on polluted and crowded highways, or merely to fret and bite our fingernails to avoid boredom and give vent to our pent-up energy."
>
> p. 2

Nelson, J. M. & L. Goldstein, "Chronic nicotine Treatment in Rats: 1. Acquisition and Performance of an Attention Task," Res. Comm. Chem. Path. Pharmacol. 5(3): 681-693, May, 1973.

> "Our conclusion is that chronic nicotine treatment does improve the efficiency of responses to goal- or incentive-related stimuli without causing or being accompanied by a generalized increase in gross activity."
>
> p. 692

Seltzer, C. C., "Effect of Smoking on Blood Pressure," American Heart Journal 87(5): 558-564, May, 1974.

> "These data suggest that cigarette smoking tends to have an inhibiting effect on blood pressure, with minimal pressure rises even instances of substantial weight gain. When this inhibiting effect of cigarette smoking is removed, as in the case of the quitters, sharp rises in blood pressure are evident."
>
> p. 563

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Ontario Perinatal Mortality Study Committee, Second Report of the Perinatal Mortality Study in Ten University Teaching Hospitals, Ontario Department of Health, Ontario, Canada, 1967. (TI) (SH)

> "There was no evidence that smoking was associated with a higher incidence of congenital malformations. There was no indication, either, of higher perinatal mortality among infants born to mothers who smoked."
> p. 28

National Academy of Sciences, Committee on Maternal Nutrition, Maternal Nutrition and the Course of Pregnancy, National Academy of Sciences, Washington, D. C., 1970, 241 pp. (SH)

> ". . . smoking is not significantly associated with excess fetal or neonatal mortality or the incidence of congenital malformation."
> p. 14

Yerushalmy, J., "The Relationship of Parents' Cigarette Smoking to Outcome of Pregnancy Implications as to the Problem of Inferring Causation From Observed Associations," American Journal of Epidemiology 93(6): 443-456, June, 1971. (SH)

> ". . . neonatal mortality rate and the risk of congenital anomalies of low-birth-weight infants were considerably lower for smoking than for nonsmoking mothers."
> p. 443

Yerushalmy, J., "Cigarette Smoking and Low-Birth-Weight Babies: Reply to Mr. Goldstein," American Journal of Obstetrics and Gynecology 114(4): 571-573, October 15, 1972. (SH)

> "The hard scientific data show conclusively that it is not reasonable to expect that giving up smoking will cause a rise in birth weight."
> p. 573

Yerushalmy, J., "Effects of Smoking on Offspring," Contemporary Obstetrics and Gynecology 1(5): 13-15, May, 1973. (TI) (SH)

> "Smoking may be considered an index that characterizes the smoker, but smoking, per se, is only incidental as a causal factor in the observed phenomena. In short, the difference in incidence of LBW (low-birth-weight) may be due to the smoker not the smoking."
>
> p. 14

> "This evidence . . . suggests that antismoking efforts will not lead to a rise in the average birthweight of infants nor to a lowering of the perinatal mortality rate. Indeed, it is important to consider that efforts to persuade women to stop smoking during pregnancy because of the alleged threat to their infants may produce undesirable guilt feelings and emotional stress in those women who find it impossible to stop."
>
> p. 15

Yerushalmy, J., "Congenital Heart Disease and Maternal Smoking Habits," Nature 242: 262, March 23, 1973. (TI) (SH)

> "We found no difference in the proportion of smokers and non-smokers between mothers of affected and unaffected children. Thus we found no difference between the children of smoking and non-smoking mothers with regard to incidence of congenital heart disease."
>
> ". . . the mortality rate is not greater for infants of smokers, and the perinatal mortality rate of low birth weight infants of smoking mothers is significantly lower than that of low birth weight infants of non-smoking mothers."

James, W. H., "Smoking in Pregnancy," Nature 246: 235, November 23, 1973. (SH)

> "It seems likely that smokers, in general, lead a markedly different life-style from non-smokers, and that the life-style of the smoking mother during pregnancy may be less supportive of the developing fetus. So it is proper to be cautious in interpreting the association between maternal smoking and perinatal death."

Burch, P.R.J., "Smoking and Pregnancy," Nature, p. 177, November 16, 1973. (TI) (SH)

> "This collective evidence therefore fails to corroborate the causal hypothesis. Each of its features is remarkably consistent with the view that the smoker, rather than the smoking, is responsible for the high incidence of low birth weight infants."

Editorial, "Smoking, Pregnancy and Publicity," Nature 245: 61, September 14, 1973. (SH)

> "The woman who can give up smoking easily is a different type of person from the one who cannot, and for all we know may be less prone to perinatal fatality and light babies. There is a danger in all statistical studies in moving from parameters to the physical world. . . . We can likewise infer that those who gave up smoking on average improved their children's health prospects but we cannot, from statistics, infer how or even that smoking is the key to it.
>
> "Mothers-to-be have always been under pressure to avoid excessive weight gains and this pressure, it is well known, frequently causes distress. Cigarettes often keep both weight and nerves under control-- it is quite possible that advice to stop smoking may have exactly the wrong effect on the mother's total health."

Hickey, R.J., et al., "Smoking Hazards to the Fetus," British Medical Journal 3: 501, 552, September 8, 1973. (SH)

> ". . . 'the smoking behaviour of women and the birth weights of their children are influenced by a common cause--the individual genotype' or constitution."
>
> p. 501

Burch, P.R.J., "Smoking, Pregnancy and Publicity," Nature, p. 245, October 5, 1973. (SH)

> ". . . the best-surviving low-birth-weight infants were born of couples in which the wife smoked and the husband did not: the most vulnerable were produced by couples in which the wife did not smoke and the husband did."

Allen, Lt. H.B., et al., "Smokers Wrinkles?" Journal of the American Medical Association 229: 1067-1069, August 27, 1973. (TI)(SH)

> "On the basis of clinical and histologic examinations, wrinkles in the 'crow's foot' area were shown to be caused by actinic exposure, not by cigarette smoking. By including black patients in our study, the factor of sunlight exposure was effectively controlled; in these patients (smokers and nonsmokers), facial wrinkles were absent.
>
> p. 1067

> "The 'prime' cause for wrinkling in exposed skin is actinic radiation. . . . Smoking plays no important role in the development of wrinkled skin in the 'crow's foot' area or elsewhere."
>
> p. 1068

Hardy, J. B., "Birth Weight and Subsequent Physical and Intellectual Development," New England Journal of Medicine, pp. 973-974, November 1, 1973. (TI) (SH)

> "Factors such as low socioeconomic status, extremes of maternal age, short stature, cigarette smoking, the presence of certain pathologic states, and low maternal weight gain during pregnancy all adversely affect birth weight. . . . Among 32 factors affecting birth examined simultaneously . . . the amount of weight gained by the mother during pregnancy and her prepregnant weight showed the strongest correlations with the weight of the infant at birth. . . ."
>
> p. 973

> "We failed [Hardy & Mellits, Lancet 2:1332-1336, 1972] to demonstrate statistically significant differences in physical growth and intellectual performance of seven-year-old children of women who had smoked 10 or more cigarettes per day throughout pregnancy as compared with those of women who had not smoked."
>
> p. 974

Pettersson, F., et al., "Perinatal Mortality," Acta Paediatrica Scandinavica 62(3): 221-230, May 1973.

> "Babies of primiparae with breech deliveries and of smoking mothers showed lower perinatal mortality rates than children of corresponding contrasted mothers. . . ."
>
> pp.228-229

Johnstone, F. & L. Inglis, "Familial Trends in Low Birth Weight," British Medical Journal 3: 659-661, 1974.

> ". . .when those patients who were known not to smoke were studied the sisters of the mothers of light-for-date babies group still had lighter babies than the other groups combined and this was also true of those patients who were known to smoke."
>
> p. 660

> "In conclusion, our findings do offer support for the Ounsted theory. There can be no doubt that sisters of women who have produced a light-for-date baby tend to have babies with relatively reduced intrauterine growth. This tendency is independent of social class, maternal size, or smoking habits."
>
> p. 661

Miller, H. C., & K. Hassanein, "Maternal Factors in 'Fetally Malnourished' Black Newborn Infants," American Journal Obstetrics & Gynecology 118(1): 62-67, 1974.

> "Maternal factors significantly associated with 'fetal malnutrition' included a poor maternal weight gain, little or no prenatal care, pre-eclampsia, and chronic major illness. Maternal factors that were not sigificantly associated with 'fetal malnutrition' were smoking, being on welfare, being unmarried, obesity, underweight, maternal age, and number of school years completed."
>
> p. 62

> "Preliminary results suggest that the lower birth weights of infants born to smoking mothers are associated with a shorter body length of the infants and not to reduced weight-length ratios."
>
> p. 67

Goldstein, H. & P. J. Wedge, "The British National Child Development Study," World Health Statistics Report 28(5): 202-212, 1975.

> "There still remains the question, however, of whether the association with birth weight and mortality is causal or simply a consequence of a link with a third factor, such as the constitution of the mother. As we have already said, it is ultimately impossible to resolve this through epidemiological studies alone."
> p. 209

Reckzeh, G., et al., "Testing of Cigarette Smoke Inhalation for Teratogenicity in Rats," Toxicology 4: 289-295, 1975.

> "No significant differences were found for litter weights, litter sizes, length of foetuses, number of implantation sites and the incidence of resorptions in utero between smoke-exposed and control mothers. . . . There is . . .no significant statistical evidence of malformations in human newborns whose mothers smoked during pregnancy."
> p. 294

Yaglou, C.P., "Ventilation Requirements for Cigarette Smoke," Transactions of the American Society of Heating and Air-Conditioning Engineers 61: 25-32, 1955. (SH)

> "The carbon monoxide concentration was much too small to affect the nonsmoker, even at the lowest air flow of 5 cfm per smoker, when the room was filled with bluish smoke."
>
> p. 31

Stewart, R.D., et al., "Experimental Human Exposure to Carbon Monoxide," Archives of Environmental Health 21(2): 154-163, August 1970. (SH)

> "The most important finding was that an eight hour exposure to 100 ppm of CO, resulting in a COHb saturation of 11% to 13%, produced no impairment of performance in the tests studied in this select, healthy group of volunteers. The tests chosen for investigation were those felt to be of practical significance in the performance of vocational endeavors and of automobile driving. . . ."
>
> p. 163

Harke, H.P., "The Problem of Passive Smoking" (Zum problem des "passiv-rauchens"), Munchener Medizinisch Wochenschrift 51: 2328-2334, December 18, 1970. (Translation) (SH)

> "This experimental study gives evidence that other reports on the assimilation of smoke compounds in 'passive' smokers seem to be based on estimations only and do not correspond with the real situation."
>
> p. 2328

> "In these investigations, we have attempted to determine to what degree the nonsmoker could be endangered by his presence in a smoke-filled room. The experiments show that under 'normal' conditions, with respect to the amount of smoke supplied versus the amount of ventilation, the nicotine content and the carbon monoxide content of room air lie distinctly under the MAK-values [maximum work place concentration]."
>
> P. 2331

Decision No. 79032, Case No. 9138, Before the Public Utilities Commission of the State of California, San Francisco, California, August 10, 1971. (SH)

> "There was no testimony that the average non-smoker's health is impaired by exposure to the smoke produced by a nearby smoker."

Health Aspects of Smoking in Transport Aircraft. Joint Study, Federal Aviation Administration, Department of Transportation and National Institute of Occupational Safety and Health, Department of Health, Education and Welfare, December 1971. (SH)

> ". . . it is concluded that inhalation of the by-products from tobacco smoke generated as a result of passengers smoking aboard commercial aircraft does not represent a significant health hazard to nonsmoking passengers."
>
> p. 45

Interstate Commerce Commission, No. MC-C-6748, Smoking by Passengers and Operating Personnel on Interstate Buses, Decided November 8, 1971. (SH)

> "We agree with the examiner's conclusions that petitioner has failed adequately to demonstrate the deleterious effects of second-hand smoke upon the health of motorbus passengers."
>
> p. 264

O'Donnell, R.D., et al., "Low Level Carbon Monoxide Exposure and Human Psychomotor Performance," Toxicology & Applied Pharmacology 18: 593-602, 1971. (SH)

> "It is concluded that the present data do not support the hypothesis that low level carbon monoxide exposure of humans results in performance decrement."
>
> p. 593

Harke, H.P. & A. Bleichert, "The Problem of Passive Smoking (Zum problem des passivrauchens)," Int. Arch. Arbeitsmed. 29: 312-322, 1972. (Translation) (SH)

> "The quantities of smoke absorbed during passive smoking are too small to cause a significant change in the skin temperature of nonsmokers, even when the nonsmokers are located in rooms containing extremely large smoke concentrations."

Harke, H. P., et al., "Passive Smoking: Concentration of Smoke Constituents in the Air of Large and Small Rooms as a Function of Number of Cigarettes Smoked and Time," Int. Arch. Arbeitsmed. 23(4): 323-339, 1972.

> "It must be emphasized at this point that it does not appear possible that such smoke densities can be generated by smokers.
>
> "This experiment using smokers [rather than smoking machines] would only be reproducible when 50 persons were placed in the room (floor space 62m^2), and each of them would smoke three cigarettes during a period of 30 minutes. The irritation . . . would be so great that certainly a large majority of the smokers would have to leave the room prior to the end of the smoking period."

Echardt, R.E., et al., "The Biological Effect From Long-Term Exposure of Primates to Carbon Monoxide," Archives of Environmental Health 25(6): 381-387, December 1972. (SH)

> "The conclusion is reached that these levels of carboxyhemoglobin for two years did not lead to any biologically significant changes in the cynomolgus monkey."
>
> p. 381

Lightfoot, N.F., "Chronic Carbon Monoxide Exposure," Proceeding Royal Society of Medicine 65(9): 798-799, 1972. (SH)

> "Decrements in visual discrimination in flicker fusion frequency, in time interval estimation tests, and difficulties in fine limb coordination and the performance of complex tasks have been reported at levels of 2-10% carboxyhaemoglobin. . . . Much of this evidence is disputed. . . . There is no evidence to show differences in performance between smokers, who may have up to 15% carboxyhaemoglobin levels. . . . Workers who have failed to take account of this may have contributed to the conflicting results reported at seemingly similar carboxyhaemoglobin levels."
>
> p. 798

Bridge, D. P. & M. Corn, "Contribution to the Assessment of Exposure of Nonsmokers to Air Pollution from Cigarette and Cigar Smoke in Occupied Spaces," Environmental Research 5: 192-209, 1972. (SH)

> ". . . out results suggest that concentrations of CO from cigarette and cigar smoking do not present an inhalation hazard to nonsmokers."
>
> p. 208

Stewart, R.D., et al., "Effect of Carbon Monoxide on Time Perception," Archives of Environmental Health 27(3): 155-60, September 1973. (SH)

> "The results of these time perception studies indicate that the acute exposure of healthy adults to concentrations of CO up to 500 ppm, which result in COHb saturations as great as 20%, has no detrimental effect on man's time sense. Thus the studies coroborate the previously reported investigation of Stewart, et al., and O'Donnell, et al."
>
> p. 159

Schievelbein, H., "On The Question of the Effect of Tobacco Smoke on The Morbidity of Non-Smokers," Internist 14(5): 236-243, 1973.

> "No proof of a threat to the health of non-smokers through 'passive smoking' can be found in studies available to date."

Stewart, R. D., et al., "Carboxyhemoglobin Concentrations in Blood from Donors in Chicago, Milwaukee, New York, and Los Angeles," Science 182: 1362-1364, December 28, 1973.

> "Among the nonsmokers, 76 percent in Los Angeles, 74 percent in Chicago, 35 percent in New York, and 26 percent in Milwaukee had COHb saturations in excess of 1.5 percent, indicating CO exposure in excess of that permitted by air quality standards."
>
> p. 845

Stewart, R. D., "The Effects of Low Concentrations of Carbon Monoxide in Man," Chapter 3.7 in Environmental Tobacco Smoke Effects on the Nonsmoker (R. Rylander, Ed.) Scandinavian Journal of Respiratory Diseases, Supplement 91: 56-62, 1974.

> "Compliance with the current U.S.A. Ambient Air Quality Standards for CO (8.7 ppm for 8 hours or 35 ppm for 1 hour), should protect everyone except those with near-terminal cardiovascular or lung disease, states which require oxygen-enriched environments. Adequately ventilated interior rooms in which tobacco smoking is permitted, should have CO concentrations no greater than those permitted by the Air Quality Standards and hence, should pose no health problem from a COHb standpoint to persons exposed to environmental tobacco smoke."
>
> p. 61

Rylander, R., "Workshop Results," Chapter 4 in Environmental Tobacco Smoke Effects on the Nonsmoker (R. Rylander, Ed.) Scandinavian Journal of Respiratory Diseases, Supplement 91: 56-62, 1974.

> "In view of the above, a personal conclusion is that the risk for the development of chronic pulmonary effects due to environmental tobacco smoke exposure is non-existent among the population in general."
>
> p. 85

Hinds, W. C. & M. W. First, "Concentrations of Nicotine and Tobacco Smoke in Public Places," New England Journal of Medicine 292(16): 844-845, April 17, 1975.

> "The data collected during this study suggest that although tobacco-smoke concentrations often exceed the annual average air quality standard for clean air, these levels would not be expected to produce the strong public reaction to tobacco smoke that has developed in the past few years. This observation suggests that annoyance from tobacco smoke is caused by factors other than the average concentration of particulate matter in the indoor atmosphere."
>
> p. 845

Huber, G.L., "Smoking and Nonsmokers--What is the Issue?" New England Journal of Medicine 292(16): 858-859, April 16, 1975.

> "Potential health effects of tobacco on the nonsmoker have recently been reviewed. . . .No data are available to demonstrate health effects of physiologic responses to nicotine levels reached in adult nonsmokers, and carbon monoxide concentrations in nonsmokers are far below levels that are of known health hazard. Potential effects of other smoke components on nonsmokers are conjectural. Information is lacking on cumulative effects of prolonged passive exposure to tobacco-smoke products."
>
> p. 859

Hinds, W.C. & M.W. First, "Smoke and Heat," New England Journal of Medicine 293(1): 48, July 3, 1975.

"The purpose of our study was not to determine if tobacco smoke is annoying, but to ascertain the particulate concentration of tobacco smoke in a variety of public places and to assess the health implications of these concentrations. Our study is in contrast to previous studies conducted under artificial conditions of greater crowding, higher smoking rate and lower ventilation rate than was found in the public places we surveyed.

It is important to keep in mind the distinction between health effects and annoyance. Unpleasant odors may be very annoying but have very little effect on health except on a psychogenic basis. It seems likely that irritating gases, unpleasant odors, peak concentrations, and high visibility play a part in the annoyance reaction to tobacco smoke."

Berkson, J., "Smoking and Lung Cancer: Some Observations on Two Recent Reports," Journal of the American Statistical Association 53(281): 28-38, March, 1958.

> "Cancer is a biological, not a statistical problem."
>
> p. 32

Langston, H. T., "The Thorax, Pleura and Lungs," Chapter 19 in Christopher's Textbook of Surgery, L. Davis, Ed., W. B. Saunders Co., (Philadelphia, 1968).

> "The evidence incriminating cigarettes in that report [Smoking and Health] came from statistical surveys. Whereas the statistical correlations may show an association between heavy cigarette smoking and the occurrence of lung cancer, clinical facets of the disease strongly dispute the cigarette's role as etiologic agent."
>
> p. 482

Carr, D., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 849-858.

> "Unfortunately, many supposedly well informed officials in the PHS and certain voluntary health organizations have permitted their emotionalism and zeal to out-distance the actual scientific knowledge and proof. This has resulted in misleading the public into believing there is proof where none exists."
>
> p. 851

Rosenblatt, M. b., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 1255-1263.

> "The widely publicized accusations of hundreds of thousands of deaths caused by cigarettes, and of shortening life expectancy a specific number of minutes per cigarette smoked are fanciful extrapolations and not factual data."
>
> p. 1256

Rosenblatt, M. B., "The Increase in Lung Cancer: Epidemic or Artifact?" Medical Counterpoint 1: 29-39, March, 1969. (CTR) (SH)

> "The prodigious increase in lung cancer during the past three decades is not due to the exposure of the population to an alleged carcinogeon but is the natural consequence of the widespread use of diagnostic techniques not previously available. The intense interest in lung cancer has also produced a tendency toward overdiagnosis of the disease on the basis of radiologic, biopsy, and cytologic findings which are often not substantiated by autopsy.
>
> p. 38

> "Since the latter part of the 19th century, there has been a progressive increase in the percentage of cases of lung cancer diagnosed prior to autopsy."
>
> p. 31

> "It would have been impossible to diagnose the unautopsied cases a few decades ago, and the increase in lung cancer is, therefore, largely dependent on diagnostic progress. . . . The progressive decline in the rate of increase suggests that the image of a lung cancer epidemic is an illusion."
>
> p. 32

Lees, T. W., "Association Between Smoking and Disease," Presented to the Standing Committee on Health, Welfare & Social Affairs, House of Commons, Ottawa, Canada, May 12, 1969. (TI) (SH)

> "In the context of smoking and disease, association has been confused with causation. That a statistical association might be found between these two factors has never been disputed. But the decision whether such an association is causative or non-causative is a separate act of judgment which must be based on medical evidence - clinical, pathological, or experimental."
>
> p. 2

Rosenblatt, M.B., et. al., "Validity of Lung Cancer Mortality Data," Bulletin of the New York Academy of Medicine, 45(6): 519-527, June, 1969. (SH)

> "It is of considerable significance that in only 40.4% of the cases the diagnosis of bronchogenic carcinoma was confirmed at autopsy and in 59.6% of the cases it was not. It is apparent, therefore, that bronchogenic carcinoma was greatly overdiagnosed during a decade (1958 to 1968) in which interest in the disease was at a high peak. If these results are representative of the findings in most general hospitals, and there is every reason to assume that they are, then the actual lung cancer mortality in the United States is distinctly less than half of the official figures. The majority of the cases certified as lung cancer are in actuality cases of pulmonary metastases from extrathoracic occult malignancies, predominantly carcinomas."
>
> pp. 524-525

Alvarez, C., "Some Statistical Practices We All Should Know," Modern Medicine, pp. 63-65, June 29, 1970. (SH)

> "It is a terribly dangerous thing for a man to start a statistical bit of research with either a strong conviction as to how things will come out, or a great reluctance to reach an unpleasant conclusion."
>
> p. 65

Ludwig, E. G. & J.C. Collette, "Some Misuses of Health Statistics," Journal of the American Medical Association, 216(3): 493-499, April 19, 1971. (SH)

> "The quality of research appears to be rather strongly related to the purposes for which the data are intended and the nature of the sponsoring agency. Data used for propaganda often suffer from most, if not all, of the fallacies we have described. Typically they are incomplete, based upon inadequate sampling, and do not relate to a general body of knowledge on the subject."
>
> p. 499

2024301259

Hickey, R.J., "Air Pollution," Chapter 9 in: Environment-Resources, Pollution & Society, Sinauer Assoc., 1971, pp. 189-212. (SH)

> "The evidence implicating smoking, particularly cigarette smoking, as a cause of lung cancer is based primarily on statistical evidence. . . . Since statistics are heavily involved, one might inquire whether the statistics have been interpreted with the rigorous objectivity demanded by science. Too often, unfortunately, when statistics are used in a problem which has some 'moral' overtones (some religions proscribe tobacco use; puritanism is skeptical of pleasure), biased, subjective interpretations may not be far behind."
>
> p. 206

Oster, K. A., "Predisposition to Atherosclerosis," Journal of the American Medical Association 222(6): 704, November 6, 1972. (SH)

> "The danger of these epidemiological studies is that they try to get much yield from little effort. It is time, in my opinion, that different approaches to the prevention of atherosclerosis are tried, especially by the NHLI, instead of juggling stale and insufficient data."

Hickey, R., "Environmental Chemical Mutagens: Are They Health Hazards?" (Excerpt printed in Ecolibrium), Ecolibrium 1(1): 12, June, 1972. (SH)

> "It is a much too common part of scientific methodology these days, in dose-response experiments in biology, to employ unrealistically high doses. . .of some chemical at varying levels in studies on small numbers of inbred experimental animals. It is also commonplace to extrapolate high level findings into quite low dosage regions in which it is quite difficult to detect effects, and to assert on the basis of subjective judgment or opinion something about the effects in this low dosage region and what it means to human health. How much regulation or law has been made by such speculation I will leave to you to estimate. Based on my understanding of science and scientific methods, extrapolation into unknown regions is scientifically invalid, and should be called by its proper name: speculation."

Schievelbein, H., "On the Question of the Effect of Tobacco Smoke on the Morbidity of Non-Smokers," Internist 14(5): 22, 1973. (Translation).

> "It goes without saying that speculations and conclusions based on speculations have no room in a scientific report. It appears necessary to say this, because rarely has there been more speculation in any area of medicine than in that of 'smoking and health.'"

Rosenblatt, M. B., et al., "Diagnostic Accuracy in Cancer as Determined by Post Mortem Examination," Progress in Clinical Cancer 5: 71-80, 1973.

> "The under diagnosis of most carcinomas sharply contrasts with the over diagnosis of bronchogenic carcinoma. The reasons for the latter vary but, in general, they are the result of renewed interest in the disease, diagnostic enthusiasm and reliance on techniques that are not infallible."
>
> p. 76-77

> ". . .the epidemic increase is based chiefly on medical certification and not on autopsy findings. When our findings were compared with those obtained in the late 19th century and early 20th century, it is found that the autopsy incidence of lung cancers was higher in those eras."
>
> p. 77

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag (Stockholm, 1973) pp. 341-362.

> "Too much statistical efforts and too little biological common sense have been applied in most cases leading to statistically valid results of little biological meaning."
>
> p. 350

> ". . .lack of exactness and piercing thought cannot be compensated for by sophisticated statistics."
>
> p. 358

Feinstein, A.R. & C.K. Wills, "Cigarette Smoking and Lung Cancer: The Problems of 'Detection Bias' in Epidemiologic Rates of Disease," Clinical Research 22(3): 555, April, 1974. (CTR) (SH)

> "'Detection bias' can distort the statistical data of a cause-effect association if a disease that sometimes escapes detection is diagnostically sought with more vigor in patients exposed to the alleged cause than in patients without such exposure. Since many lung cancers are not diagnosed during life, detection bias might create a falsely high association with cigarette smoking if smokers were particularly likely to receive diagnostic tests for identifying the cancer.
>
> "These results, which suggest that the current increase of lung cancer in women may arise mainly from improved detection, also evoke suspicions that cigarette smoking may lead more to the diagnosis of lung cancer than to the disease itself."

Burch, P.R.J., "Smoking and Coronary Heart Disease (Cont.)," New England Journal of Medicine 290(6): 345, February 7, 1974. (SH)

> "To attribute mortality differences between doctors and the general male population to changes in smoking habits--in the absence of any quantitative analysis of cause and effect--is to replace scientific method by wishful thinking."

Burch, P.R.J., "Smoking and Lung Cancer: Burch's Reply," New Scientist, p. 559, February 28, 1974. (SH)

> "Expositions of the causal hypothesis suffer, I submit, from two outstanding weaknesses. They rely heavily on qualitative statements unsupported - and sometimes contradicted - by quantitative evidence. Second, no mechanism of tobacco carcinogenesis has been proposed that can be tested quantitatively."

2024301262

Oser, B. L., "The Misuse of Scientific Data," Paper Presented at the AIC/AAAS Symposium on "Responsible Use and Misuse of Scientific Data," January 26, 1975. (Abstract Only)

> "Scientific data are generally assumed to findings, generally expressed in numerical terms, from which inferences may be drawn. The validity of the interpretation of scientific data to establish 'facts' depends upon the design of the investigation, the reliability of the observation, and the competence and integrity of the investigator, or whoever is responsible for the interpretation. To represent scientific data as ultimate truth ignores the subjective aspects of both the investigation and the inferences drawn from it.
>
> "Properly used, scientific data not only add to our store of information, but generate new hypotheses and concepts which may further advance the frontiers of science. Misused, whether by laymen or by scientists themselves, such data may perpetuate ignorance, cause confusion and ultimately impair confidence in, and support of, science."

Burch, P.R.J., "Smoking and Lung Cancer: Burch's Reply," New Scientist, p. 559, February 28, 1975.

> "Expositions of the causal hypothesis suffer, I submit, from two outstanding weaknesses. They rely heavily on qualitative statements unsupported - and sometimes contradicted - by quantitative evidence. Second, no mechanism of tobacco carcinogenesis has been proposed that can be tested quantitatively."

Huber, G.L., "Smoke and Heat," New England Journal of Medicine 298(1): 48-49, July 3, 1975.

> "Controversy continues to surround many issues related to smoking. When scientific data on the effects of an agent on health are incomplete, as they are on the tobacco question, reactions in many people are derived far too often from an emotional rather than an objective basis. I should like to make a plea as a partisan for objective science. Emotional arguments with a moral flavor, presented without scientifically acceptable data, have, in my judgment, no place for solving problems as serious as this one.
>
> "In other words, results or conclusions should not be presented or interpreted with a preconceived bias of the investigator or, for that matter, of the reader. Unfortunately, for reasons I cannot fully understand, this course has far too often been followed in questions of tobacco and health. Rather, definitive answers should be obtained by careful scientific endeavors designed to test in an objective and honest manner a clearly delineated hypothesis."
>
> p. 48

Seltzer, C.C., "Smoking and Cardiovascular Disease," American Heart Journal 90(1): 125-126, July, 1975.

> "The history of medicine throughout the centuries contains many examples of evangelical fervor for etiologic or therapeutic theories that were later shown to be wrong. A prime responsibility of epidemiologists is to maintain the skepticism of science amidst the passions of evangelism. If smoking is related to CHD in only a limited segment of the population, the people who are not at risk will hardly be benefited by blunderbuss interventions aimed at everyone. If smoking is not causally related to CHD, the true situation will never be discerned unless investigators observe the cardinal scientific principle of ruling out counter-hypotheses. Until conclusive proof is available, the health of the public and the welfare of science demand a balanced consideration of all the available evidence."

Sterling, T.D., "A Critical Reassessment of the Evidence Bearing on Smoking as the Cause of Lung Cancer," American Journal of Public Health 65(9): 939-953, September, 1975.

". . .the belief that smoking is a major cause of lung cancer still lacks definitive experimental demonstration but depends almost exclusively on the result of statistical surveys. The designs and execution of these surveys have been severely criticized (as well as hotly defended) in the past, and the discovery that the antecedents of lung cancer are found in many alternative and interactive causes may again create the need to reevaluate the results of these epidemiological studies."

p. 939

"If we pull together the information which has become available in the last few years about the prospective studies, we find substantial support for the possibility that the findings linking smoking to lung cancer, and perhaps also to other diseases, were due to a faulty selection process that introduced a large number of biases."

pp. 945-946

"The readiness with which the existing evidence has been accepted as demonstrating causality for cigarette smoking perhaps is the best measure for the desire to keep our world simple and orderly. But cancer is a complex disease."

p. 949

Eskwith, I.S., "Etiology of Atherosclerotic Heart Disease," American Heart Journal 90(6): 809-810, December, 1975.

"Over the years, physicians have assumed many diseases to be caused by tobacco. Had they been true, the cities and towns in America today would be desolate. Buffalo would once more be roaming the plains and the rivers would be again teaming with the great numbers of fish that Henry Hudson viewed in the river named for him. Furthermore, the Japanese who respond as we do to tuberculosis, pneumonia and carcinoma, indulge in a high rate of cigarette smoking but have a low incidence of coronary artery disease."

p. 809

Bair, W.J., et al., "Apparatus for Direct Inhalation of Cigarette Smoke by Dogs," Journal of Applied Physiology 26(6): 847-850, June 1969. (SH)

> "Most of these experiments, such as exposing animals to cigarette smoke in a smoke chamber or introducing cigarette smoke to the anesthetized animal through a tracheotomy aperture, lack similarity to human smoking habits. The validity of extrapolating results from such experiments to possible effects in man is therefore highly questionable."
>
> p. 847

Editorial, "The Cigarette-Cancer Dispute," The New York Times, May 9, 1970. (SH)

> "Scientific truth is determined ultimately by open examination of data and full discussion by all competent persons in a field. The Hammond-Auerbach results should be scrutinized intensively by all interested parties."

Brower, L.P., "Smoking & Cancer," The New York Times, February 15, 1970. (SH)

> "All this experiment proves is: Whether or not smoking causes lung cancer in beagles is as inconclusive now as it was before the experiment was carried out."

Buhler, V.B., Letter to Hon. Tim Lee Carter, Congressional Record, E2639, March 26, 1970. (SH)

> ". . . the unpublished report by Dr. Auerbach must be viewed with considerable restraint. I sincerely hope that his reporting of 'early squamous cell bronchial carcinoma' in only two dogs will not be viewed by your Committee as scientific proof as to whether or not cigarette smoking has been established as the cause of lung cancer in humans."

Sterling, T.D., "Comment on Smoking Dogs," Archives of Environmental Health 22: 631-32, May 1971. (SH)

> "The need for providing adequate controls, especially in experiments that involve surgical incision and bypassing of natural functions, is so deeply engrained in biological science that its violation in this important piece of work defies comprehension. Indeed, the consequences of this work will be confusion rather than enlightenment."
>
> pp. 631-32

Buhler, V.B., Letter to Hon. Tim Lee Carter, Congressional Record, E1856, March 15, 1971. (SH)

> "My previous concern about this study has been confirmed by the finally published article, which so completely fails to bear out the claims announced at the American Cancer Society's press conference last February."

Hickey, R., "Environmental Chemical Mutagens: Are They Health Hazards?" (Excerpt printed in Ecolibrium) Ecolibrium 1(1): 12, June 1972. (SH)

> "It is a much too common part of scientific methodology these days, in dose-response experiments in biology, to employ unrealistically high doses . . . of some chemical at varying levels in studies on small numbers of inbred experimental animals. It is also commonplace to extrapolate high level findings into quite low dosage regions in which it is quite difficult to detect effects, and to assert on the basis of subjective judgment or opinion something about the effects in this low dosage region and what it means to human health. How much regulation or law has been made by such speculation I will leave to you to estimate. Based on my understanding of science and scientific methods, extrapolation into unknown regions is scientifically invalid, and should be called by its proper name: speculation."

Editorial, Environment 15(4): 21, May, 1973.

> "The report was first rejected by the Journal of the American Medical Association, and on at least two subsequent occasions, independent pathologists have reviewed Dr. Auerbach's records and found no evidence of cancer.
>
> "Much of this research, like that of Dr. Auerbach, has been supported by the American Cancer Society, which has so far refused to allow examination of the raw data produced in its extensive epidemiological studies."

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag, Stockholm 1973, pp. 341-362.

> "The animal models used to study the development of arteriosclerosis, and in particular coronary arteriosclerosis, ischaemic heart disease and myocardial infarction are not representative of the human disease. Consequently, though much work has been invested in these animal experiments few conclusions can be drawn from the results of these to the clinical or human situation. This is especially true for the huge literature on dietary studies using maximally distorted diets with nonrealistic relations when compared with ordinary clinical situations. This is also true for animal studies on behaviour, cigarette smoking (or influence of nicotine or of carbon monoxide)."
>
> p. 350

Hickey, R. J., et al., "Cigarette Smoke As A Carcinogen?" American Review of Respiratory Disease 111: 105-106, 1975.

> "In studies in which experimental animals were exposed to realistic levels of tobacco smoke, the results have generally failed to support the hypothesis that smoking causes lung cancer."
>
> p. 150

Greene, S. N., Statement, Hearings Before the House Committee on Interstate and Foreign Commerce, June 30, 1964.

> ". . .the inference that tobacco smoking is a cause of lung cancer is not based on experimental evidence but on statistical judgment, and. . .causation cannot be inferred from statistical association. . . . Experimental confirmation is essential. . .but to date this has not been forthcoming, and the nature of the relationship of tobacco smoking to lung cancer remains an unresolved question."

Burford, T. H., Statement, Hearings Before the House Committee on Interstate and Foreign Commerce, June 30, 1964.

> "I do not believe that lung cancer is caused by cigarette smoking and I do not believe smoking is responsible for any shortening of life. The oft-quoted statistics that smoking does cause lung cancer or does shorten human life have done nothing more than perhaps establish certain statistical associations which fall far short of proving a causal connection."

Kissen, D.M., "Possible Contribution of the Psychosomatic Approach to Prevention of Lung Cancer," Medical Officer, pp. 343-345, December 24, 1965. (SH)

> ". . . those with a poor outlet for emotional discharge appear to have more than four and a half times the mortality rate for lung cancer compared with those with a good outlet, and more than two and a half times the rate of those with a moderate outlet."

Fiorentino, M., "Lung Cancer in the U.S.: Observations on the Age at Death," Medical Record and Annuals, 61(7): 228-230, 1968. (SH)

> "An examination of the trends of age at death, however, brings up some points which do not seem to have an immediate and clear explanation consistent with the theory of association [of cigarette smoking and lung cancer], often called causation."
>
> p. 228

Cooper, D.A., et al., "Primary Carcinoma of the Lung in Nonsmokers," Archives of Enviromental Health, 16(3): 398-400, March 1968. (SH)

> "A considerable amount of literature based upon epidemiologic, statistical and experimental studies relates smoking to lung cancer, although the finite cause remains unknown. Because the concentration on the effects of tobacco smoke may obscure the search for other factors involved in the etiology of lung cancer, we report on 63 cases of lung cancer occurring in nonsmokers from a series of 1,372 cases of proven primary carcinoma of the lung. . . ."
>
> p. 398

Langston, H.T., "The Thorax, Pleura and Lungs," Chapter 19 in Christopher's Textbook of Surgery, L. Davis, (Ed.), W. B. Saunders Co., (Philadelphia, 1968). (SH)

> "The evidence incriminating cigarettes in that report [Smoking and Health] came from statistical surveys. Whereas the statistical correlations may show an association between heavy cigarette smoking and the occurrence of lung cancer, clinical facets of the disease strongly dispute the cigarette's role as etiologic agent."
>
> p. 482

Kotin, P., Testimony, Hearings Before a Subcommittee of the Committee on Appropriations, House of Representatives, 1968, pp. 663-687 (SH)

> "I, as a pathologist, cannot look at a lung and say damage to lung tissue is due to cigarette smoke."
>
> p. 686

Brem, T.H., Testimony, Hearing Before the Committee on Interstate and Foreign Commerce, U.S. House of Representatives, April 15-May 1, 1969, pp. 1063-1076. (SH)

> ". . . only a very small proportion (probably less than 2 percent) of heavy smokers over many years develops cancer of the lung. The incidence is even much lower for smoking women. If smoking were indeed an important cause of lung cancer, it is difficult to explain how 98 percent of smokers of long duration escape the disease. Again, logic dictates that there must be something very different about these 2 percent, other than their smoking habits. This difference has, of course, not been identified as to its nature."
>
> p. 1068

Buhler, V.B., Testimony, Hearings Before the Committee on Interstate and Foreign Commerce, U.S. House of Representatives, April 15-May 1, 1969, pp. 769-787. (SH)

> "The disease seems to appear in smokers, non-smokers, light smokers and heavy smokers at about the same age. Also, the average age is reported to be advancing. People seem to have taken up smoking at earlier ages over the last 30 years. Yet the median age at death from lung cancer for white males in 1949 was reported to be age 61 and in 1965 the median age has increased to nearly age 65. If cigarette smoking causes lung cancer, why doesn't it occur sooner in those who start smoking early in life?"
>
> pp. 771-772

Fisher, R.H., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 1214-1216. (SH)

> "If cigarettes were the cause of lung cancer, I believe we would have an incidence many times greater than we do now, and would not encounter the disease in non-smokers."
>
> p. 1216

Rigdon, R.H., "Cigarette Smoking and Lung Cancer: A Consideration of This Relationship," Southern Medical Journal, 62(2): 232-235, 1969. (SH)

> "A statistical association between cigarette smoking and lung cancer has been demonstrated; however, such an association does not constitute a 'cause and effect' relationship. Many scientists question this association. Experimental attempts have failed to support this statistical association."
>
> p. 235

Lees, T. W., "Association Between Smoking and Disease," Presented to the Standing Committee on Health, Welfare, & Social Affairs, House of Commons, Ottawa, Canada, May 12, 1969. (TI) (SH)

"No good evidence has been produced to show that the accepted association between smoking and lung cancer and many other diseases is one of cause and effect."

p. 16

"Behind the assertion that smoking causes cancer lies the implication that the meteoric rise of lung cancer is unique in the history of cancer so it must have some unique specific external 'cause.' This is not the case. Taking both sexes together, the death rate from cancers of the upper alimentary tract (mouth + gullet + stomach) was nearly as great in England forty years ago as lung cancer is today yet they are now in rapid decline. No one suggested that these cancers must have a single external dominant cause."

p. 17

"I suggest that the rise of lung cancer in Europe and North America in the middle of the 20th century is largely a natural phenomenon. This rise can be more accurately and plausibly fitted to a general theory of the occurrence of specific diseases than to the theory that its dominant cause is an undetermined chemical in cigarette smoke."

p. 27

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Malhotra, S.L., "Clues to the Possible Mode of Action of Cigarette Smoke in the Pathogenesis of Lung Cancer," Journal of the Indian Medical Association 55(8): 265-270, October 16, 1970. (SH)

> ". . . there still remain many unsolved problems in the aetiology of carcinoma of the lung which tend to obscure the cigarette hypothesis."
>
> p. 265

Rosenblatt, M.B., et al., "Causes of Death in 1,000 Consecutive Autopsies," New York State Journal of Medicine 71(18): 2189-2193, September 15, 1971. (SH)

> "Carcinoma of the lung was the only neoplasm which was greatly overdiagnosed clinically and in which no unsuspected cases were found at autopsy."
>
> p. 2192

Rosenblatt, M.B., et al., "Prevalence of Lung Cancer: Disparity Between Clinical and Autopsy Certification," Medical Counterpoint, pp. 58-59, October 1971. (SH)

> "There was a marked contrast in accuracy in the diagnosis of lung cancer as compared with other internal neoplasms. Autopsy confirmation in carcinomas of the colon, pancreas, stomach and ovary was very high whereas in carcinoma of the lung the diagnosis was verified in only 45 per cent of the cases."
>
> p. 58

McCall, M.G. & N.S. Stenhouse, "Deaths from Lung Cancer in Australia," The Medical Journal of Australia pp. 524-525, March 6, 1971. (SH)

> "The effect of an environmental agent such as air pollution would be strongly supported by evidence that the death rate from lung cancer in British immigrants increases with increasing periods of residence in England before immigration to Australia. . . . Since smoking habits do not vary greatly between the countries studied, the findings reported here strongly support Dean's conclusion that the role of air pollution in the genesis of lung cancer has been seriously underestimated."
>
> p. 525

Feinstein, A.R., "Neoplasms of the Lung," in Cecil-Loeb Textbook of Medicine, Beeson, P.B. & W. McDermott, Eds., W. B. Saunders Co, (Philadelphia, 1971). (SH)

> "No single cause for lung cancer has been identified. . . . The many conflicting claims and counterclaims about the cause of lung cancer will probably not be resolved until prolonged, well-designed clinical epidemiologic studies can be conducted."
>
> p. 924

Kutty, M.K. & M. Balasegaram, "Malignant Tumours in West Malaysia," Journal of the Royal College of Surgeons Edinburgh 17(2): 102-107, 1972. (SH)

> ". . . there was no significant correlation between smoking and lung cancer in our series. Although Malays, like the Chinese, indulge in smoking, the disparity in the incidence does not support the theory that smoking play an important role in the aetiology of lung cancer."
>
> p. 106

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Herrold, K. McD., "Survey of Histologic Types of Primary Lung Cancer in U.S. Veterans," Pathology Annual, 7: 45-79, 1972. (SH)

> "Extremely important from a biologic standpoint is that only a small percentage of heavy cigarette smokers develop lung cancer."
>
> p. 74

> "There was no correlation found between the histologic type of primary lung cancer and the amount of tobacco smoked among the 'current smoker of cigarettes only.'"
>
> p. 77

Sterling, T.D. & S.V. Pollack, "The Incidence of Lung Cancer in the U.S. Since 1955 in Relation to the Etiology of the Disease," American Journal of Public Health, pp. 152-58, February 1972. (CTR)(SH)

> "There is unequivocal evidence of an 'urban' factor for lung cancer, as distinct from smoking patterns or questions of classifications or diagnosis. This excess in urban areas for lung cancer decreases by size of city when smoking patterns are held constant, and varies by geographical areas. The native white male mortality rate for lung cancer is over 100% greater in urban areas than in rural areas. . . .
>
> "The fact that the incidence of lung cancer is leveling off at a time when it ought to have increased (if smoking is the major cause of lung cancer) ought to give us some pause. Together with other anomalies, these data suggest the possibility that particulate pollution rather than smoking may be the primary source of the incidence of lung cancer in the United States."
>
> p. 157

Langston, H.T., "Lung Cancer-Future Projection," Journal of Thoracic and Cardiovascular Surgery 63(3): 412-415, March 1972. (SH)

> "Based on age incidence studies of lung cancer for the 30 year period from 1939 to 1968 at the Veterans Administration Hospital, Hines, Illinois, the following comments are justified: 1. The currently recognized wave of increased incidence in lung cancer is principally composed of persons born between 1890 and 1900. 2. When this generation passes on there should be a marked reduction in the overall problem of lung cancer in this institution. This is to be expected by about 1980."
>
> p. 415

Lewin, R., "Towards Perfect Man," New Scientist, pp. 38-39, October 4, 1973. (SH)

> "He [Burch] has looked at lung cancer too and concludes that smoking is not causal. He points out that when cigarette smoking first became popular in 1900 it was with men only. Women took up the habit some 30 years later. This being so, he claims, one would expect to see kinks in the incidence of lung cancer at different stages for men and women since the start of the century. There are no kinks."
>
> p. 39

Langston, H.T., "Swimming Up Stream," Grant Physician 1(2): 10-11, July 1974. (SH)

> "In putting all of this together, several points could be clearly stated that helped me decide against accepting the 'popular' view of a causal relation between tobacco and lung cancer.
>
> 1. Lung cancer is predominantly a disease of males.
> 2. It has a very clearly defined age incidence. The peak occurs between 55-70 years in the general population.
> 3. There is no clearly established dosage factor. Victims of lung cancer who started smoking at an early age and smoked heavily developed their lesions in the same average age period as the other lung cancer victims.
> 4. German pathologists, notably Von Kikuth, who illustrated his material by a graph, showed this disease to be increasing in frequency in autopsy material beginning around 1890 to 1900. This was long before cigarettes became popular."
>
> p. 10